SIR: Charlton (1995) presents a point of view which I understand as follows: traditional diagnostic categories should be abandoned because they lack explanatory power, and should be replaced by diagnoses in terms of deficits to information processing modules (analogous to software packages, either applications or operating systems). Unfortunately he does not pay any attention to the *act* of diagnosis in the real clinical world: that is, a transaction between an individual patient and an individual psychiatrist in a specified social setting, which includes the patient's family and the psychiatrist's professional context. The act of diagnosis occurs when an individual and his/her (often invisible) family decide to approach a doctor with a problem with living, which may include (for example) interpersonal hostility, role failure as parent or partner, or difficulties at work or school. The patient and his/her social network approach the doctor for an explanation, but primarily to seek a solution to the difficulties in their lives. The solution which is offered or accepted will be embedded in the explanation.

I would argue that in offering explanations, we should consider not merely scientific questions of reliability and validity, but also questions which may loosely be characterised as philosophical. What bearing does the explanation have on the individual's sense of self, of volition and agency? How does the explanation influence the broader network's sense of responsibility? What claims does the explanation make on absolute truth and to what extent does it demand unquestioning belief, or invite refutation and the generation of alternatives? How does it influence the nature of the relationship between doctor and patient? The function of explanation as narrative, subject to revision and elaboration in an interpersonal context, has been articulated by, for example, Pocock (1995): the central idea is that explanation (or diagnosis) has the power to do both harm and good in a therapeutic setting, regardless of its scientific status.

I do not question the scientific accuracy of Charlton's 'PC model' but I question the utility of this kind of reductionism in the real world of human suffering, and the absence of regard for the interpersonal nature of the diagnostic process. The term 'schizophrenic' has been subject to criticism, but I would rather be a schizophrenic than a personal computer.

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SIR: Charlton (1995) is right to draw our attention to the need for psychatrists to develop "a cognitive nosology based upon a modern understanding of human psychological architecture". He argues, and I agree with him, that our present classificatory systems lack explanatory power and he therefore calls for the use of single case studies of patients with defined cognitive lesions but he fails to give examples of which psychiatric cases would count as "pure psychiatric cases". He also proposes a 'PC' model of the mind which harbours an homunculus.

I believe that the emphasis in the new research agenda should not be placed on some such notion as a 'pure case' but rather on symptoms such as verbal hallucinations, visual hallucinations, particular types of delusions, formal thought disorder, thought insertion, thought withdrawal, etc. The sites responsible for these symptoms and the psychological and neurophysiological processes underlying these symptoms should become the focus of study. In addition, it is clear that there may be psychopathological processes which unify symptoms which appear on a superficial inquiry to be dissimilar. For example, a good case can already be made for saying that verbal hallucinations and some kinds of passivity experiences are different manifestations of a similar if not identical neuropsychological impairment, namely errors in the monitoring of intentional acts. It is equally important to emphasise that there are numerous psychotic symptoms which psychatrists have not been taught to attend to or recognise, particularly in this current intellectual climate where a menu approach to psychiatric diagnosis has become prominent. These symptoms include such symptoms as paraprosopia (Ellis *et al*, 1994) and 'forced gaze', a symptom which is too readily classified as a passivity experience without the implications for visual processing being taken into account.

Charlton's description of a 'PC' model of the mind must be understood only as an analogy. Computer technology has given us a new language and also new conceptual tools for thinking about the mind. However, Charlton errs when he "assumes that the 'conscious mind' behaves like a computer operator who is inspecting a continuous printout in order to generate appropriate behaviours". This way of conceiving of the mind falls into

the category fallacy which Gilbert Ryle (1949) described as the 'dogma of the Ghost in the Machine'. There is no empirical evidence of a single cortical area which all other cortical areas report exclusively to either in the visual or in any other system (Zeki, 1993). This suggests both that the brain must be using a different strategy for generating integrated mental experiences (i.e. solving the binding problem) and for answering the question of *who* is looking at a visual image. The current hypothesis, which is in itself inadequate, is that our awareness of our mental experiences as an integrated whole is the result of the synchronised firing of all the neurones symbolising all the different attributes of, for example, a single object (shape, colour, movement, etc) (Crick, 1994). In other words, our unified perceptual experiences do not depend upon an homunculus.

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Lead-in placebo washout period

SIR: A reviewer of the multi-centre risperidone trial report pointed out the dubious utility of the one week lead-in washout period (Johnson & Johnson, 1995). This is an important issue that merits attention. The principal purpose of the washout period is to metabolise and eliminate the previous medication. As the reviewer points out, and as available data demonstrate (Cohen *et al*, 1988), one week is far too short to accomplish that goal. Nevertheless, sudden discontinuation of the previous treatment for several days may result in clinical deterioration; in this risperidone trial, the deterioration was severe enough to necessitate a shortened washout in 17% of the patients (Peuskens, 1995). Delaying treatment or withdrawing it has ethical and economical costs. If the principal purpose of the washout period is not achieved, why incur these costs?

To answer this question, it may be suggested that a partial washout is better than none. But I am not sure that this is self-evident. Another possible justification is that the washout period allows the establishment of the "true baseline" (Kane *et al*, 1994). But it is not clear what the "true baseline" means. If it means psychopathology in an untreated state, this would not apply to patients who have been receiving treatment until a week ago and whose brains still contain substantial amounts of the medication. Furthermore, placebo washout period might be justified as a method to screen for and eliminate placebo responders. But data on placebo responders eliminated from antipsychotic trials are hard to find. Finally, one might say that the washout is needed to eliminate the effects of the street drugs. But this purpose could be met in other ways, without withdrawing or delaying antipsychotic medication.

The washout period was introduced decades ago; at that time, pharmacokinetic data were not available, and ethical as well as economical concerns were different. The merits of the washout were questioned and an alternative method using an initial low-dose haloperidol treatment period was suggested (Hirsch & Barnes, 1990). Nevertheless, the washout period continues to be a standard component of antipsychotic trials. It is time to reconsider its justification.

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Obstetric complications in schizophrenia

SIR: The interesting findings of the British Perinatal Mortality Survey study by Sacker *et al* (1995) which found an excess of obstetric complications (OCs) in